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MIDTERM RESULTS OF THE ROTATION-ADVANCEMENT FLAP METHOD FOR CORRECTION OF PARTIAL ANOMALOUS PULMONARY VENOUS DRAINAGE INTO THE SUPERIOR VENA CAVA

Hiroshi Kubota, MD[a]
Akira Furuse, MD[a]
Yutaka Kotsuka, MD[a]
Kuniyoshi Yagyu, MD[a]
Kazuhiko Hirata, MD[a]
Yuji Murakawa, MD[b]

Since 1986, a rotation-advancement flap method has been used in 11 patients with partial anomalous pulmonary venous drainage into the superior vena cava. This method consists of atrial partitioning, enlargement of the superior vena cava, and protection of the sinus node. The midterm postoperative sinus node function and hemodynamic changes were examined in this study. Postoperative angiograms showed normal pulmonary venous pathway and no stenosis in the superior vena cava. Cardiac rhythm was normal and no clinical symptoms appeared. (J Thorac Cardiovasc Surg 1996;112:1-7)

In 1956, Kirklin, Ellis, and Wood[1] described a technique to repair partial anomalous pulmonary venous drainage (PAPVD) into the superior vena cava (SVC). The principal complications after the repair of this anomaly have been obstruction of the SVC and various types of atrial arrhythmia. In 1990, Okabe and associates[2] described in this Journal a new technique called the rotation-advancement flap method to obviate these complications (Fig. 1). The early results of postoperative electrophysiologic and hemodynamic examinations in three patients were also reported to be satisfactory. The purpose of this article is to report the midterm evaluation of this procedure.

From the Department of Cardiothoracic Surgery,[a] Second Department of Internal Medicine,[b] University of Tokyo, Tokyo, Japan.

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Address for reprints: Hiroshi Kubota, MD, Department of Cardiothoracic Surgery, Tokyo University, 7-3-1, Hongo, Bunkyo-ku, Tokyo, Japan, 113.

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Patients and methods

Since 1986, 11 patients, 6 male and 5 female, with PAPVD into the SVC underwent successful surgical correction by the rotation-advancement flap method as described in the previous report.[2] This method consists of atrial partitioning with a polytetrafluoroethylene sheet, enlargement of the cavoatrial junction by a right atrial flap, and protection of the sinus node. All of the patients are alive and well. Follow-up hemodynamic and electrophysiologic studies were done on seven patients, from whom informed consent was obtained, between 11 months and 7 years, 5 months after the operation (Table I). The PAPVD was diagnosed by cardiac catheterization in six patients. In one case (case 5), the diagnosis was made intraoperatively although cardiac catheterization and

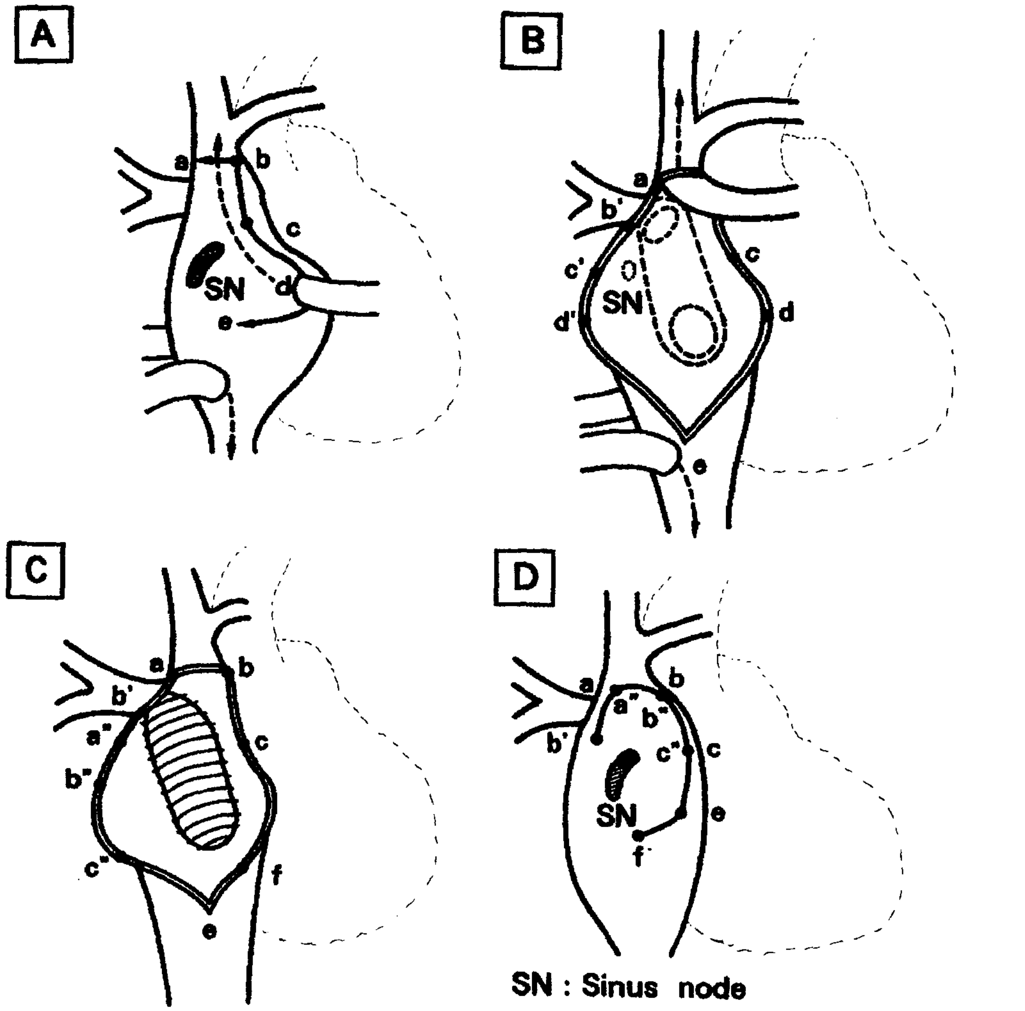

Fig. 1. **A,** Incision is made from *a* to *e*. **B,** When original atrial septal defect is small, intraatrial septum is resected to create large atrial septal defect. **C,** Sheet of 0.2 mm polytetrafluoroethylene is sutured so that anomalous pulmonary venous blood is directed into left atrium. **D,** Right atrial wall is used as flap to widen atriocaval channel. Points *a-a"*, *b-b"*, and *c-c"* are approximated for rotation-advancement of flap.

Table I. *Patient profiles*

Case No.	*Age at op. (yr)*	*Sex*	*Postop. period (yr)*	*Diagnosis*	*Associated anomaly*	*Preop. rhythm*
1	23	F	7.4	PAPVD	PFO, PLSVC	SR
2	6	M	7.3	PAPVD	ASD (SV), PLSVC	SR
3	7	F	6.8	PAPVD	ASD (SV)	SR
4	4	F	3.0	PAPVD	ASD (SV)	SR
5	44	M	3.0	PAPVD	ASD (SV)	SR
6	6	F	2.8	PAPVD	ASD (SV), PLSVC	SR
7	11	M	0.9	PAPVD	PFO, PLSVC	SR

F, Female; *PFO,* patent foramen ovale; *PLSVC,* persistent left SVC; *ASD,* atrial septal defect; *SV,* sinus venosus type; *SR,* sinus rhythm.

echocardiography were done preoperatively. The mean follow-up time was 4 years, 6 months. Patient ages ranged from 6 to 44 years.

Five patients had an atrial septal defect, all of which were the sinus venosus type. Two patients had no atrial septal defect but a patent foramen ovale. Four patients had persistent left SVC. Preoperative cardiac rhythm was regular sinus rhythm in all patients. As a postoperative hemodynamic measurement, the pressure gradient between the SVC and the right atrium was measured by catheterization. Superior vena cavograms and pulmonary arteriograms were obtained. The diameters of the cavoatrial junction and the SVC were measured.

Electrocardiogram and Holter electrocardiogram were recorded. As postoperative electrophysiologic studies, sinus node function and atrioventricular (AV) conduction were evaluated. The corrected sinus node recovery time was derived by subtracting the resting sinus cycle length from the maximum sinus node recovery time. The sinoatrial conduction time was derived by the method of Narula.[3] Corrected sinus node recovery time and sinoatrial conduction time were measured both immediately and long term after operation. Observed intrinsic heart rate, defined as the rate of spontaneous depolarization of the sinus node independent of the influences of the autonomic nervous system, was determined by a modification of the protocol of Jose.[4] Propranolol, 0.2 mg/kg of body weight, was administered intravenously at a rate of 1 mg/min. Ten minutes thereafter 0.04 mg/kg of atropine sulfate was administered in a single injection over 2 minutes. The maximum sinus rate after atropine administration was taken as the observed intrinsic heart rate. The ratio of observed intrinsic heart rate (IHRo) to the lowest it could be (IHRo/[IHRpredicted[5] − 2 SD*]) was taken as a quantitative measure of the integrity of intrinsic sinus node function. By this method, a ratio of 1.0 or greater was an indication of normal sinus node function (intrinsic heart rate index[6]). The earliest activation site of the right atrium was assessed by catheter mapping. Three 5F quadripolar catheters were inserted from the right femoral vein: two of them were fixed at the point of the high right atrium and His bundle and one was steered to determine the earliest activation site of the right atrium. The Wenckebach point of AV node was determined under various rates of rapid atrial pacing. Among the studied patients, three were reluctant to undertake the cardiac catheterization, so that angiographic and electrophysiologic data were not obtained for them. Because one patient had atrial fibrillation even before operation, he was excluded from this study.

Statistical evaluation. Values for data are presented as mean plus or minus the standard deviation. The sinoatrial conduction time and the corrected sinus node recovery time were compared by paired *t* test. A *p* value of less than 0.05 was considered statistically significant.

Results

Hemodynamic measurements. There were no significant pressure gradients between the SVC and the right atrium (Table II). The mean pressure in the SVC was 6.0 ± 2.8 mm Hg. The mean pressure in the right atrium was 5.9 ± 2.6 mm Hg. The new atriocaval tunnel was found to be adequate in size by frontal and sagittal cavograms in all patients (Fig. 2, *A* and *B*). The mean diameter of the atriocaval junction was 13.8 ± 4.2 mm. The values were the same as or larger than the diameter of the SVC (11.1 ± 3.2 mm). The pulmonary venous return was also normal in the angiographic evaluation. The right upper (or upper and middle) pulmonary venous drainage was found successfully rerouted to the left atrium without stenosis in every case (Fig. 3).

*Standard deviations.

Table II. *Hemodynamic data*

	Pressure (mm Hg)		*Diameter (mm)*	
Case No.	*SVC*	*RA*	*SVC*	*CAJ*
1	5	5	14.0	18.0
2	9	8	15.0	18.0
3	2	2	8.5	11.0
4	10	10	10.0	13.0
5	4	4	10.0	10.0
6	7	6	6.5	8.5
7	5	6	14.0	18.0
Mean ± SD	6.0 ± 2.8	5.9 ± 2.6	11.1 ± 3.2	13.8 ± 4.2

RA, Right atrium; *CAJ,* cavoatrial junction; *SD,* standard deviation.

Electrocardiographic studies. On Holter electrocardiogram, all cases showed stable regular sinus rhythm. Case 5 showed a lower heart rate than others, although the results of the sinus node function test were within normal range. Few premature ventricular contractions were observed and premature atrial contractions could not be detected in any patients. AV block, bundle branch block, and ST segment changes were not detected. In all cases, including cases of children, no periods of junctional rhythm were found on electrocardiogram. The mean axis of postoperative P waves shifted leftward (41 ± 23 degrees before operation to −21 ± 55 degrees after operation as shown in Table III).

Electrophysiologic studies. Immediately after operation, the mean sinoatrial conduction time was 164.8 ± 40.0 msec and the mean corrected sinus node recovery time was 290.0 ± 122.1 msec. Both values were within normal limits. Midterm follow-up study also showed normal values of mean sinoatrial conduction time (122 ± 28 msec) and mean corrected sinus node recovery time (254 ± 207 msec). In case 1, although the sinoatrial conduction time immediately after the operation was prolonged (220 msec), it became normal (170 msec) after 7.4 years (Fig. 4). The observed intrinsic heart rates ranged from 70 to 92 beats/min. The mean intrinsic heart rate index was 0.95 ± 0.06. This was slightly lower than normal. The Wenckebach points of the AV

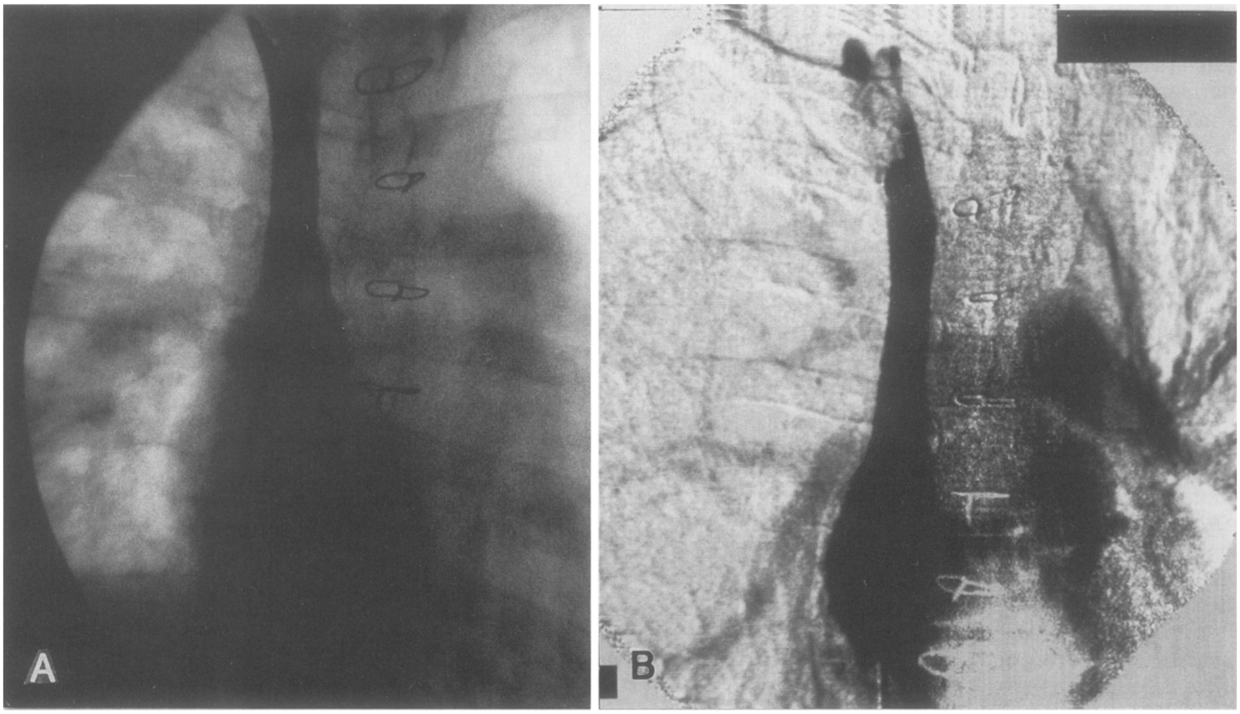

Fig. 2. A, Cavogram early after operation (case 1). SVC was not stenosed and its diameter was normal. **B,** Cavogram late (7.4 years) after operation (case 1). Diameter of SVC was normal.

Fig. 3. Pulmonary angiogram late after operation showed normal pulmonary venous return, although body surface area markedly increased in this case (case 3).

node ranged from 110 to more than 200/min (Table IV). The earliest activation sites existed in the sinus node area or perinodal area in all cases except for one in which it was located in the low right atrium (Fig. 5).

Discussion

One of the principal complications after the repair of PAPVD into the SVC is obstruction of the SVC or new intra-atrial tunnel of anomalous pulmonary venous drainage. Especially the diameter of the right SVC in patients with persistent left SVC tends to narrow. Atrial arrhythmias are other important postoperative complications. Many surgical techniques have been developed in an effort to prevent these problems. Since Kirklin, Ellis, and Wood[1] described a technique to repair this anomaly in 1956, various surgical procedures have been reported. The procedure of Kirklin, Ellis, and Wood[1] involved covering both defect and veins with a patch that directed blood flow of the anomalous pulmonary veins to the left atrium through an atrial septal defect. Schuster, Gross, and Colodny[7] recommended enlargement of the SVC with a separate patch of pericardium. Trusler and associates[8] reported late results in 29 patients who were treated with the procedure described herein. According to this report, there was slight narrowing of the SVC in eight children and a significant obstruction of the SVC in one child. Holter monitoring detected rhythm abnormalities in 13 of them. One patient with first-degree AV block also had a brief episode of ventricular tachycardia. Another had intermittent

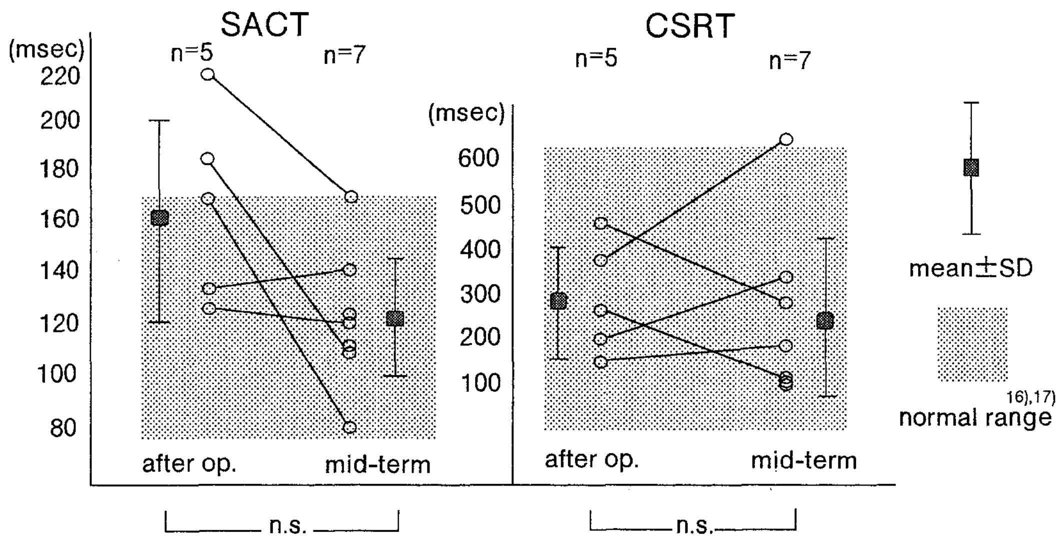

Fig. 4. Electrophysiologic data. *SACT,* Sinoatrial conduction time; *CSRT,* corrected sinus node recovery time; *SD,* standard deviation; *n.s.,* not significant.

Table III. *Electrocardiographic data*

	Heart rate (beats/min)			*Arrhythmia (No./day)*		*P wave axis (degrees)*	
Case No.	*Max*	*Min*	*Mean*	*PAC*	*PVC*	*Preop.*	*Postop.*
1	109	48	63	0	98	15	−8
2	106	73	88	0	2	40	0
3	114	70	82	0	0	70	12
4	109	60	80	0	2	25	−39
5	84	34	46	0	0	68	30
6	142	70	91	0	3	50	−135
7	147	50	86	0	0	19	−4
Mean ± SD	116 ± 22	58 ± 16	77 ± 16			41 ± 23	−21 ± 55

PAC, Premature atrial contraction; *PVC,* Premature ventricular contraction; *SD,* standard deviation.

Table IV. *Electrophysiologic data*

	SACT (msec)		*CSRT (msec)*				
Case No.	*After op.*	*Midterm*	*After op.*	*Midterm*	*IHRo (beats/min)*	*IHR index*	*Wenckebach point (beats/min)*
1	220	170	380	680	92	1.07	130
2	167	80	200	320	90	0.95	≥200
3	183	110	260	117	85	0.90	160
4	124	120	160	161	92	0.94	≥200
5	130	140	450	287	70	0.93	110
6	NS	120	NS	112	89	0.92	170
7	NS	110	NS	100	NS	NS	180
Mean ± SD	165 ± 40	121 ± 28	290 ± 122	254 ± 207	83 ± 8	0.95 ± 0.06	

SACT, Sinoatrial conduction time; *CSRT,* corrected sinus node recovery time; *IHRo,* observed intrinsic heart rate; *NS,* not studied; *SD,* standard deviation.

first-degree AV block. Other arrhythmias included sinus bradycardia (4 patients), junctional rhythm (4 patients), isolated premature ventricular contractions (2 patients), and premature ventricular contractions associated with low atrial ectopic beats (1 patient). However, the sinus node recovery time was normal in all 12 patients tested.

Puig-Massana and associates[9] used an atrial baffle to create an intracardiac tunnel diverting the anomalous venous drainage to the left atrium. The anterior wall of the heart was reconstructed with a pericardial patch. Chartland and associates[10] described a partitioning method with a longitudinal suture starting above the highest pulmonary vein

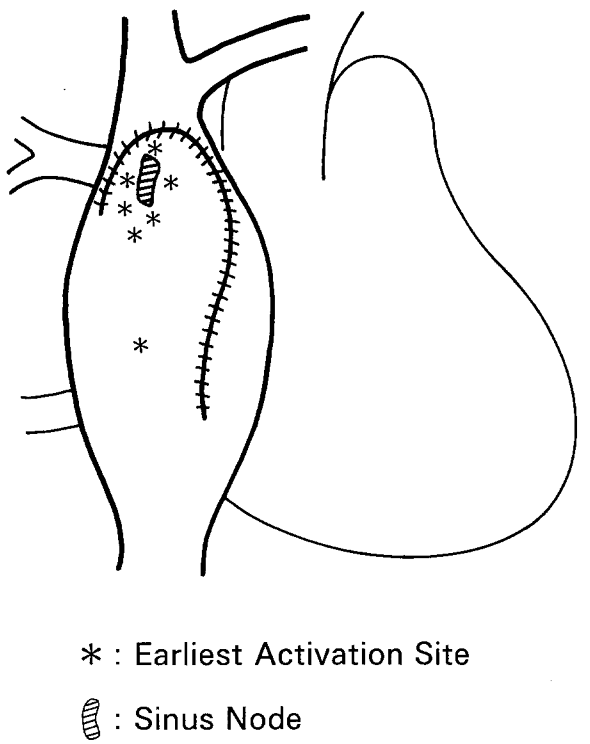

Fig. 5. Earliest activation sites of endocardial right atrial map.

directing the pulmonary venous flow through the enlarged atrial septal defect into the left atrium. The anterior cavoatrial tunnel was enlarged with a right atrial appendage–SVC angioplasty. Ehrenhaft, Theilen, and Lawrence[11] described an atrioseptopexy to shunt the pulmonary venous return through the atrial septal defect and transplantation of the SVC into the right atrial appendage. Williams and associates[12] and Warden and associates[13] described satisfactory results of modified atrioseptopexy and transplantation of the SVC into the right atrial appendage. Waldhausen and Pierce[14] described an operative procedure similar to ours with use of the atrial flap in 1985. They used a patch of autogenous pericardium to close the defect and the incision on the SVC was longitudinal.

Our report dealt with a midterm evaluation of seven cases with PAPVD treated by the previously described rotation-advancement flap method. Clinically, all patients were free of symptoms and none had an audible cardiac murmur. The SVC was not stenosed and its diameter was normally preserved. Although in some cases body surface area increased significantly with growth, the size of the new pulmonary venous channel created by a sheet of polytetrafluoroethylene was adequate. In this operative method, there is a possibility of injuring the precaval sinus node artery originating from right coronary artery. Busquet and associates[15] described the origin of the sinus node artery from the right coronary system (66%) or left coronary system (30%) and showed that double supply was infrequent (4%). Variability was found in the course of the nodal artery relative to the cavoatrial junction. Precaval artery was more frequent (58%) than retrocaval artery (36%). Therefore it is reasonable to assume that a significant number of patients had precaval sinus node artery that originated from the right coronary artery. However, this study showed that the sinoatrial conduction time and corrected sinus node recovery time were not depressed after operation. The Holter electrocardiogram showed stable regular sinus rhythm in all cases. Because the earliest activation site was detected in the high right atrium in six of seven patients, we assumed that a functioning pacemaker existed within or close to the inherent sinus node. However, no direct evidence was available to confirm this assumption. Also, it is not clear why the intrinsic heart rate was slightly lower than expected. Consequently, there is a possibility that the surgical procedures caused some effect on sinus node function. Nevertheless, because the decrease of the intrinsic heart rate was small, we considered that the function of the atrial pacemaker after the operation was sufficient for daily activity.

In cases in which the sinus node is fed by retrocaval sinus node artery originating from the right or left coronary arteries, the node will not be jeopardized by this surgical method. Although the reason patients with right precaval sinus node artery showed almost normal postoperative sinus node function is unclear, this may be related to the collateral blood supply for the sinus node from contralateral coronary arteries. The mean axis of P waves shifted leftward after the operation. Because the earliest activation sites were considered to exist in the sinus node or the perinodal area by catheter mapping, the reason of shift of the P wave axis might not indicate the shift of the pacemaker but the changed direction of the conducting wave front in the right atrial wall as a result of incision and rotation of the atrium.

Case 5 may need special considerations, because the earliest activation site in this patient existed in the low right atrium and the Holter electrocardiogram revealed chronologic incompetence although sinoatrial conduction time and corrected sinus node recovery time were within the normal range. Also notable is that this patient had a reduced Wenckebach point, indicating slightly impaired AV conduction, and that the age of the patient was the oldest in this series. Long-lasting right atrial distention as a result of atrial septal defect might have had adverse effects on the sinoatrial system even before operation. In this report, three patients with sinus rhythm did not undergo the cardiac catheterization, the ambulatory electrocardiographic recordings showed no substantial rhythm disturbance in these subjects. Although the present study is not free from the limitation caused by the exclusion of these subjects, we believe the rotation-advancement flap method successfully rerouted partial anomalous pulmonary venous drainage and reconstructed the cavoatrial junction without stenosis and without clinically significant atrial pacemaker dysfunction. The outcome of this procedure in the long term may require further follow-up.

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